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CLINICAL NEUROLOGICAL INDICATORS ARE ONLY MODERATELY CORRELATED WITH QUANTITATIVE NEUROPSYCHOLOGICAL TEST SCORES IN PATIENTS WHO DISPLAY MILD-MODERATE BRAIN IMPAIRMENT FOLLOWING CLOSED-HEAD INJURIES [1]

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Summary.—Quantitative comparisons were completed between mean scores for standardized neuropsychological tests (requiring 6 hr.), numbers of neurological signs (tests requiring 15 min.), anomalies within the electroencephalogram, subjective symptoms during a structured interview (requiring 20 min.), and an indicator of an organic brain syndrome for 81 patients who had sustained significant mechanical impacts to their skulls. The composite of neuropsychological scores was correlated 0.70 with a composite of neurological indicators and EEG anomalies. There were no significant correlations between neuropsychological or neurological scores and the numbers of subjective complaints, abnormal personality indicators, duration of coma or the interval of posttraumatic dysmnesia. However, the scores for the severity of the organic brain syndrome, partial complex epileptic symptoms, and subjective complaints were *inversely* correlated ($rho = -0.35$) with the duration of coma, the period of posttraumatic amnesia, and the numbers of qualitative electroencephalographic anomalies.

The realization that brain trauma, even without the loss of consciousness, can result in persistent reduction in neurocognitive proficiency and adaptability has challenged traditional medical mythology (Parker, 1990; Persinger, 1993, 1995). People who have sustained cerebral trauma sufficient to produce mild to moderately severe brain impairment (as defined by quantitative, norm-referenced and performance-based procedures) do not necessarily exhibit deficits that are discernable by the brief clinical examinations of neurologists. A plethora of very disruptive subjective experiences which range from encephalalgia to the sense of a presence occur in patients with either or both types of impairments (Persinger, 1994).

The ordinal ranking (mild to severe) of brain impairment is not necessarily synonymous with (mild to severe) brain damage. Legal and medical definitions of brain damage often emphasize the absence of brain tissue as defined by computer x-ray tomography or the occasional MRI (magnetic resonance imaging). However, the strength of the typical correlation between the ventricular enlargement (one index of the global amount of neuronal

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loss) and the performance component of the Wechsler Adult Intelligence Scale (WAIS–R) is about 0.40 (Cullum & Bigler, 1986). This indicates that only about 16% of the variance in performance intelligence which is remarkably insensitive to all but the more severe brain injury is accommodated by actual quantitative shifts in intracerebral volume.

When the patient displays "a normal" CT and neurological examination, the postincident changes in adaptability and subjective experiences are given default diagnostic labels that have included the posttraumatic syndrome, the postconcussional syndrome, and posttraumatic neurosis. The latter label connotatively implies a "psychological origin" to the complaints and implicitly reinforces the belief system that psychological phenomena are somehow independent of microstructural alterations in brain function (Parker, 1990). This traditional medical model does not take into account the fact that all behavior emerges within the spatial domain of the synapse-receptor complex. Permanent disruptions in behavior and adaptability can occur if this complex is chronically altered and does not require the induction of cystic lesions.

The theoretical purpose of the present study was to compare quantitatively the major domains of clinical observations that are employed to diagnose individuals who have sustained effects from substantial mechanical energies. The four domains are neurological indicators, norm-referenced neuropsychological tests, norm-referenced self-report symptoms, e.g., personality tests, and subjective experiences that are reported during brief interviews. The latter are important because some practicing clinicians who are involved with these patients assign the diagnosis and suggest global and sometimes permanent interventions *only* on the basis of a brief (30 min.) interview with the patient. The practical component to this study was to discern the actual *quantitative* relationship between these domains to examine the validity of qualitative arguments such as "people who report symptoms that are correlated with brain injury and who exhibit overt neurological indicators are impaired and hence cannot return to work."

Method

Subjects

A total of 81 patients (50 men; 31 women), 17 to 52 years of age (M = 32, SD = 10) who had been referred over a 5-year period for complete neuropsychological assessments served as subjects. This population displayed the following mean parameters (standard deviations in parentheses): formal education 12.0 yr. (2.7), time since incident 2.8 yr. (3.8) (Mdn = 1.5 yr.), estimated posttraumatic "amnesia" or (more accurately) dysmnesia 21 (32) days [Mdn = 3 days] and unconsciousness or coma (Mdn = 0.5 hr.; range = 0 to 1.5 mo.). The means and standard deviations for the Wechsler Adult Intelli-

gence Scale (Revised) were 89 (11) for the verbal cluster and 92 (12) for the performance cluster.

Procedure

Over a two-day period, the following measures were obtained by experienced psychometrists for each patient: agility (tap), agnosia and graphaesthesia for the left and right foot and hand, Trails A and B, Verbal Fluency (FAS) and Design Fluency, Corsi Block Spatial Span and Digit Span, Rey copy and recall (+30 min.), Tactual Performance Test (dominant hand, nondominant hand, memory, and localization), Speech-Sounds Perception and Seashore Rhythm Tests, Dichotic Word Listening Test accuracy for the left and right ears separately, color-word interference from the Stroop, Category Test, and the Conditioned Spatial Association Task. z scores were computed for each procedure, based upon normative means and standard deviations (Persinger & Richards, 1995), such that a negative z score reflected less proficiency or efficiency. The mean z score for these 31 measures as well as the Halstead-Reitan Impairment Index (Reitan & Wolfson, 1985) were calculated. Details and references for these procedures have been reported elsewhere (Persinger & Richards, 1995; Richards & Persinger, 1992). The z scores for the 31 variables were comparable to those presented in a previous study (Persinger & Richards, 1995).

Each patient had been evaluated by this researcher (20 years experience) during a 15-min. assessment. The measures (the most typical indicators of injury within the central nervous system that follow brain trauma) were (1) erratic or irregular movement during passive lateral pursuit (conjugate deviation) of both eyes to a point moving recursively across the visual field, (2) vertical eye movements, (3) ocular convergence to an approaching object and the associated accommodation (pupillary) reflex, (4) the light and consensual reflexes, (5) voluntary, repeated left-right movement of eyes, (6) voluntary ocular convergence, (7) sequential appositions of each finger to the thumb (right and left hand, three trials each), (8) lateral tongue movement and rapid protrusion of tongue, (9) repeated nose-to-finger (patient's finger from his nose to the examiner's finger, placed 0.5 m in front) for the left and the right hand with eyes either opened or closed, (10) repeated supination/pronation of hands on knees, (11) repeated alternation of lateral hand movements across the midline, (12) stance for the right and the left leg (10 sec.), eyes-opened, (13) stance for the right and the left leg (5 sec.) eyes-closed and (14) Rhomberg sign (extent of sway with eyes closed). Each measure received a score of 0, 1, 2, or 3. The total neurological score was the sum of the scores for these 24 indices. The evaluation was completed without knowledge of the other test results.

The second component of the neurological inference was an electroen-

cephalographic record. This measurement was not designed to replace the traditional 10-20 method of the neurologist (Niedermeyer & da Silva, 1987), but instead employed a montage with which complex partial epileptic-like symptoms and personality factors have been quantitatively associated (Makarec & Persinger, 1990). The record reflected bipolar measurements between the frontal (F7/F8), temporal (T3/T4), and occipital (O1/O2) regions as well as comparisons between the left and right combinations of these regions, e.g., F7/T3 vs F8/T4. During the 45-min. recording, which was always completed at the end of the first day, the patient sat in a comfortable chair within an acoustic chamber. This time of testing was selected because clinical observations have suggested that rest within a very quiet environment after 8 hr. of vigorous cognitive processing induced by the assessment facilitated disinhibition of neuroelectrical (cerebral) cortical anomalies. Each record was rated by two experienced technicians on an anomaly scale of 0 to 3. The qualitative characteristics included in the score were fast beta frequency, trains of theta rhythms during the waking state, absence of posterior alpha rhythms, unusual spiky-waves, odd-shaped patterns and epileptiform signatures; a record that contained a classic paroxysmal discharge (which occurred in 9/81 patients) was automatically given a maximum score of 3. The interobserver reliability coefficients ranged between 0.7 and 0.8.

A clinical interview, which required about 20 min., followed the Neuropsychological Symptom Checklist (1983). The total numbers of changes on the first page, which were primarily involved with sensory and motor alterations, and on the second page, which were more cognitive and affective, were calculated. For comparisons with standardized measures, the profile for the organic brain syndrome (Lachar, 1985) was calculated by averaging the T scores (standardized $M=50$ and $SD=10$) for Scales 1, 3, and 9 from the Minnesota Multiphasic Personality Inventory. The total score for the incidence, e.g., daily, once per week, of signs suggestive of complex partial epileptic-like signs, as defined by the inventory developed by Roberts, Varney, Hulbert, Paulsen, Richardson, Springer, Shepherd, Swan, Legrand, Harvey, Struchen, and Hines (1990), was obtained.

All statistical analyses involved SPSS software on VAX 4000 computer. Factor analyses (PA1, principal component) were performed for the Halstead-Reitan Index, the mean of the z scores for the 31 neuropsychological indicators, the total neurological score, the sensory-motor component of the checklist, the cognitive-affective component of the checklist, the mean T score for the organic brain syndrome, the anomaly score for the electroencephalogram, and the total score for Roberts' inventory for complex partial epileptic-like symptoms (hence only 8 composite variables were employed in the factor analysis). Multiple regression analyses were completed, using the mean z score values from the comprehensive neuropsychological examination

or the Halstead-Reitan Impairment Index (range 0 to 1) as the dependent variables and the other variables as independent variables. Partial correlations were completed to accommodate any potential confounding effects of age and education. Additional correlations (Spearman *rho* and Pearson *r*) were completed for the estimated duration of the posttraumatic dysmnesia, the duration of coma or unconsciousness, and the time since the injury with the eight major clusters that represented the four domains of inquiry: neuropsychological deficits, overt neurological indicators, subjective experiences, and inferences of personality. To ensure that some general personality factor had not been masked, a "neurosis" score, composed of the mean of the *T* scores for Scales 1, 2, and 3 from the Minnesota Multiphasic Personality Inventory, and a "psychosis" score, composed of the mean scores for Scales 6, 8, and 9 from that inventory, were calculated. These measures were correlated with the other major scores obtained in this study.

Results

The means and standard deviations for the major clusters for the 81 patients who have been referred for assessment subsequent to a closed-head injury were: mean *z* score for the neuropsychological tests −1.18 and 1.04; impairment index 0.43 and 0.28, neurological score 10.0 and 8.0, anomalous EEG rating 1.2 and 0.9, subjective sensory-motor complaints 5.2 and 2.4, subjective cognitive-emotional complaints 4.0 and 2.7, the organic brain syndrome scaled score 69 and 10, and Roberts' Total Complex Partial Epileptic-like Symptoms score 66 and 39. The results of the factor analysis after varimax rotation are shown in Table 1. Only two factors emerged. Factor 1 (eigenvalue = 2.94) explained 37% of the variance while Factor 2 (eigenvalue = 2.03) explained an additional 25% of the variance.

TABLE 1

Factor Loadings For Two Major Factors That Emerged For Scores For Neurological, Neuropsychological, Subjective Experiences, and Standardized Psychological Tests in a Population of Patients Who Sustained Closed-head Injuries

Variable	Factor 1	Factor 2
Neuropsychological *z* Score	−.15	−.91
Halstead-Reitan Index	.13	.88
Neurological Signs	.19	.72
Anomalous EEG Rating	−.39	.48
Subjective Sensorimotor Problems	.62	.15
Subjective Cognitive Deficits	.72	.20
Organic Brain Syndrome Score	.85	−.12
Roberts' Subclinical Seizure Score	.84	.12

Fig. 1 shows the scattergram between the mean of the *z* scores for the neuropsychological tests (that required 6 hr. to complete) and the total score for the neurological indicators (15 min.). The total neurological score accom-

modated about 50% of the variance of the neuropsychological tests. Multiple regression analyses, with the mean z score as the dependent variable and all other measures as predictors, resulted in a multiple R of 0.70. The variables and standardized partial regression coefficients (*beta*) according to the order in which they entered the equation were total neurological score (−0.62) and anomalous EEG rating (−0.27); the standard error of the estimate was 0.72. Because the zero-order correlation between the Halstead-Reitan Impairment Index and the mean of the z scores for the 31 measures was only −0.81, multiple regression analysis was completed for the former measure as well. However, only the same two variables entered that equation.

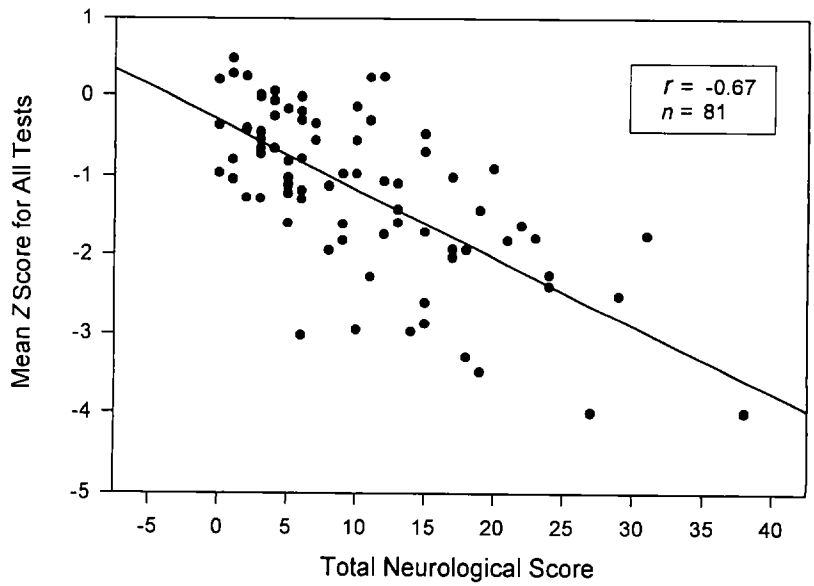

FIG. 1. Scattergram for the correlation between scores for the total neurological signs (requiring 15 min.) and the mean z score for 31 standardized neuropsychological tests (requiring 6 hr.).

When the association between age and the total neurological score was first removed, the correlations between the latter score and the mean z score for neuropsychological impairment (partial $r = -0.60$) and the Halstead-Reitan Impairment Index ($r = 0.50$) were reduced in magnitude but were still statistically significant ($p < .001$); however, the initial partial correlation for formal education did not affect the magnitude of the coefficients by more than 0.02. There were also no statistically significant differences between the sexes (all Fs < 2.00) for any of the measures (including age, duration of coma, and posttraumatic amnesia) except for the numbers of subjective sen-

sory-motor complaints. Women reported significantly ($F_{1,79}=3.65$, $p=.05$) more ($M=5.8$, $SD=2.5$) complaints of subjective-motor experiences than did the men ($M=4.7$, $SD=2.1$).

The two additional clusters of personality variables that have influenced traditional diagnosis of "neurosis" (MMPI Scales 1, 2, and 3) and "psychosis" (MMPI Scales 6, 8, and 9) showed means (and standard deviations) of 72 (12) and 63 (8), respectively. Neither cluster was significantly associated with the neuropsychological, neurological, or electroencephalographic measurements. Only the mean of the scales that comprised the "psychosis" cluster was correlated with the numbers of complex partial epileptic-like symptoms ($r=0.60$, $p<.001$) while the "neurosis" cluster was correlated with all of the subjective indices, i.e., sensorimotor complaints ($r=0.40$), cognitive complaints ($r=0.50$), and complex partial epileptic-like symptoms ($r=0.64$).

The distributions of the durations for coma/unconsciousness and the estimated duration of posttraumatic amnesia were skewed. Consequently Spearman *rho* (rank order) correlations were completed. These two measures were *negatively* correlated with the clusters of scales from the MMPI. The correlations (all $p<.01$) for these clusters with the duration of coma and posttraumatic dysmnesia, respectively, were "organic brain syndrome" −.38 and −.32; neurosis cluster −0.35 and −0.42; and total complex partial epileptic-like symptoms −0.42 and −0.30. The score for the MMPI psychosis cluster was correlated significantly only with the duration of coma (−0.34) but not with the duration of amnesia (−0.18). However, there were no statistically significant ($p>.05$) correlations for the duration of coma or posttraumatic amnesia with the Halstead-Reitan Impairment Index, our total impairment score, the numbers of neurological indicators, or an anomalous electroencephalographic record for this population.

Discussion

This study was designed to estimate the *quantitative* associations between measures of neuropsychological deficits, neurological signs, personality, and subjective experiences for patients who were described by physiatrists or neurologists as displaying mild (or occasionally) moderate brain damage or the postconcussional syndrome. The second purpose of the study was to evaluate the utility (reliability) of employing neurological indicators (obtained during a 15-min. clinical assessment) and the numbers of subjective concerns that are reported by the patient to predict standardized deficits in neurocognitive processing.

The results indicated a moderate strength of association between standardized test results and semiquantitative estimates of neurological function. The strongest prediction, which accommodated only 50% of the variance in

the mean score for standardized neuropsychological testing, was based upon the total neurological score and the severity of qualitative anomalies within electroencephalographic records. Although this concordance would be impressive epidemiologically, the standard error of the estimate was too large to rationalize the substitution of the norm-referenced neuropsychological results by the composite neurological and electroencephalographic indicators for *individual* cases except (perhaps) within the most extreme increments of the scale, e.g., <5 and >25.

The second important result from this study is that (1) the total numbers of symptoms and concerns reported by the patient, (2) elevations on scales within a personality inventory that has been employed by clinicians to infer clinical variations of the "organic brain syndrome" (as well as gross classifications such as "neurosis" or "psychosis"), and (3) the total scores for an inventory that reflects the frequency of incidence of complex partial epileptic-like experiences, were *not* significantly associated with the severity of the actual, standardized neurocognitive deficits. These results indicate that attempts to infer the presence of neurocognitive impairment strictly upon the basis of the total numbers of "complaints" during a structured "interview" may not be valid. The caveat to this conclusion is the possibility that weighted, quantitative combinations of specific symptoms may still be discriminative (Persinger, 1994).

The results of this study strongly suggest that a thorough neuropsychological assessment must include inferences of personality and subjective experiences. Scores for these domains can be abnormal (more than 2 standard deviations from the normalized mean) without being correlated with the intensity of neuropsychological deficits. The absence of any consistent association between psychiatric indicators and global neuropsychological impairment is compatible with the hypothesis that even very small alterations in neurostructural function might evoke subjective distress as severe and as incapacitating as those associated with more obvious neuropsychological or CT-verified anomalies (Persinger, 1995). Alterations in the sense of self can be as disruptive to adaptation, particularly to previous vocational and family settings, as a physical disability (Miller, 1993; Persinger, 1994).

The absence of significant correlations for these quantitative impairment indices with the duration of either coma or posttraumatic amnesia does not appear commensurate with the previous literature (Corkin, Hurt, Twitchell, Franklin, & Yin, 1987; van Zomeren & van den Burg, 1985). The most plausible explanation for this discrepancy would be the subpopulation who were referred for assessment. We would have received patients who were not conspicuously impaired or vegetative but who were exhibiting persistent problems of adaptation.

Although many patients whose initial Glasgow Coma scores were below

8 ultimately exhibit intercorrelated deficits that would be quantitatively conspicuous ($SD > 3.0$) to the neuropsychologist and qualitatively evident to the neurologist or physiatrist (Levin, 1993), the factors that determine this association may not be valid within populations of patients who sustain minor head injuries. As emphasized by Schoenhuber and Gentilini (1989), the Glasgow Coma Scale is not sufficiently sensitive to assess these patients accurately and has no prognostic value for early or late complications. To help address this potential problem the correlation between the composite neurological score and the mean z score for the quantitative tests was computed separately for those patients who reported no suspensions of consciousness ($n = 37$; 46% of sample) and those who had displayed loss of consciousness or coma for more than 24 hr. ($n = 17$; 21% of sample; all verified by inspection of medical records). The Spearman *rho* correlations for these two subpopulations were −0.70 and −0.78 (both $ps < 001$), respectively.

The results of this study clearly suggest that applying the "traditional arguments" of induced unconsciousness or persistent posttraumatic amnesia as medical or litigational proofs of impairment may not be valid (Parker, 1990). Our most recent test-retest study (Tiller & Persinger, in preparation) indicated no statistically significant changes in scores on any of the 31 standardized tests between one year and four years following a traumatic brain injury for patients whose initial mean standardized (z score) deficit averaged −2.8. This absence of change occurred in context of verbal reports of "recovery" by both the patients and their families.

The significant negative association between the qualitative anomalies within the electroencephalographic records and the total numbers of subjective complaints, scores for the organic brain syndrome and the total numbers of complex partial epileptic-like experiences reaffirms the questionable validity of the typical, *connotative* conclusion: "a normal EEG implies no significant neurocognitive disruption." The results support a previous hypothesis that these disruptive experiences may be driven by electrical anomalies within subcortical limbic (primarily hippocampal-amygdaloid) structures. With an intact electrocortical manifold that can process and organize the neuroelectrical correlates of these experiences, the patient can become more aware of their occurrence. The increased proportion of these subcortical inputs would ultimately alter the neuroelectrical configuration from which the sense of self is generated.

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